In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: July 30, 2024

S.D., a minor, by and through her parent and natural guardian, SAMANTHA DETERS,	*	PUBLISHED
Petitioner,	*	No. 19-459V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Table Injury, Measles-Mumps-Rubella ("MMR") Vaccine; Encephalitis.
Respondent.	*	

Bridget Candace McCullough, Muller Brazil, LLP, Dresher, PA, for Petitioner.
Lauren Kells, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

I. INTRODUCTION

On March 28, 2019, Samantha Deters ("Petitioner"), parent and natural guardian of S.D., a minor, filed a petition under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges S.D. suffered a

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

Table encephalitis injury as a result of a measles-mumps-rubella ("MMR") vaccination administered on April 27, 2016. Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating Petitioner did not meet "her burden of establishing entitlement to compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 27).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[3] the undersigned finds that Petitioner has proved by preponderant evidence the criteria required to establish that S.D. suffered a Table claim for encephalitis post-MMR vaccination, and therefore, she is entitled to compensation.

II. BACKGROUND

A. Procedural History

On May 28, 2019, Petitioner filed her petition along with medical records and an affidavit. Petition; Petitioner's Exhibits ("Pet. Exs.") at 1-10. Additional medical records were filed from May 2019 to January 2020. Pet. Exs. 11-15. Thereafter, this case was reassigned to the undersigned. Notice of Reassignment dated Mar. 16, 2020 (ECF No. 22).

Respondent filed his Rule 4(c) report on June 24, 2020, arguing against compensation. Resp. Rept. at 1. Petitioner filed an expert report from Dr. Frederick Nahm on March 22, 2021, followed by updated medical records in April and May 2021. Pet. Exs. 16, 26-31. Respondent filed an expert report from Dr. Max Wiznitzer on January 18, 2022. Resp. Ex. A.

Thereafter, the undersigned held a Rule 5 conference. Rule 5 Order dated Apr. 7, 2022 (ECF No. 60). The undersigned preliminarily found Petitioner would be able to prove a Table injury for encephalitis following MMR vaccination. Id. at 3. Additional expert reports were filed by both parties. Pet. Ex. 38; Resp. Ex. C.

The parties requested to resolve entitlement through a ruling on the record. Joint Status Rept., filed June 7, 2022 (ECF No. 64). Petitioner filed her motion for a ruling on the record on September 11, 2023, and Respondent filed his response on November 13, 2023. Pet. Motion for a Ruling on the Record ("Pet. Mot."), filed Sept. 11, 2023 (ECF No. 97); Resp. Response to Pet. Mot. ("Resp. Response"), filed Nov 13, 2023 (ECF No. 98).

This matter is now ripe for adjudication.

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[3] Although this Ruling does not discuss all of the medical records, expert reports, and medical literature, the undersigned reviewed and considered all of the evidence submitted in this matter. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); Simanski v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), aff'd, 601 F. App'x 982 (Fed. Cir. 2015).

B. Factual History

1. Summary of Medical Records[4]

S.D. was born on April 8, 2015. Pet. Ex. 1 at 1. Delivery was induced at 37 weeks due to intrauterine growth restriction ("IUGR"). Pet. Ex. 8 at 33. S.D. weighed five pounds at birth. Pet. Ex. 1 at 1. A newborn examination on April 13, 2015, was normal except for jaundice. Id. at 2. On April 21, 2016, S.D. had a pediatric newborn checkup with no major concerns noted. Pet. Ex. 9 at 14-15.

On June 23, 2015, at eleven weeks of age, S.D. was admitted to the hospital for a choking episode during which she turned pale, but had no loss of tone or seizure-like movements. Pet. Ex. 8 at 18-19. She was kept overnight for observation. Id. It was noted that she had an upper respiratory infection ("URI") and a possible ALTE (apparent life-threatening event). Id. During a follow-up on June 26, 2015, S.D.'s examination was normal aside from nasal congestion. Pet. Ex. 1 at 4-5.

On July 7, 2015, S.D. presented to pediatrics for a two-month well visit. Pet. Ex. 1 at 7. Her examination and development were noted to be normal. Id. At this visit, S.D. received Pentacel (diphtheria-tetanus-acellular pertussis ("DTaP")/inactivated poliovirus ("IPV")/haemophilus type b conjugate ("Hib")), Prevnar, Rotateq (rotavirus), and her second hepatitis B vaccinations. Id. at 8. Two weeks later, on July 20, 2015, S.D. returned to pediatrics for a follow-up appointment regarding poor weight gain. Id. at 9.

S.D. had a four-month well visit on September 4, 2015. Pet. Ex. 1 at 17. She was noted to be doing well developmentally. Id. She was squealing and was oriented to voice. Id. An examination was normal. Id. at 17-18. S.D. received a second round of Pentacel, Prevnar, and rotavirus vaccinations with no reported adverse reaction. Id. at 18.

S.D. had her nine-month well visit on January 20, 2016. Pet. Ex. 1 at 21. Petitioner denied any recent problems and had no developmental concerns. Id. S.D.'s developmental milestones included sitting up and saying "dada" and "mama" (non-specific). Id. An examination was normal, and she received her third round of Pentacel, hepatitis B, Prevnar, and rotavirus vaccinations and the influenza ("flu") vaccine. Id. at 21-22. No adverse reaction was noted. Id.

On February 1, 2016, S.D. saw her pediatrician for cough, nasal congestion, and fever. Pet. Ex. 1 at 26. She was diagnosed with a URI. Id. at 27. On February 26, 2016, S.D. was seen for gastroenteritis with vomiting, decreased appetite, and fussiness. Id. at 28. On April 19, 2016, S.D. was diagnosed with another URI. Id. at 32-33.

[4] This summary is largely taken from the factual summary taken from Respondent's brief, along with edits by the undersigned. See Resp. Response at 2-13.

On April 27, 2016, S.D. saw her pediatrician for a one-year well visit. Pet. Ex. 1 at 34. Developmentally, she was walking, pulling to stand, and had a thumb-finger grasp, but she had no words and was not saying "dada" and "mama" (specific). Id. An examination was otherwise normal. Id. at 34-35. S.D. was diagnosed with speech delay. Id. at 35. At this visit, MMR, varicella, hepatitis A, and meningococcal vaccines were administered. Id.

Twenty-one days later, on May 18, 2016, S.D. presented to pediatrics. Pet. Ex. 1 at 37. Petitioner had concerns that S.D. had been clumsy for about one week. Id. Petitioner also reported that S.D. had been very cranky, which she attributed to teething. Id. It was noted that S.D. had started walking three months earlier and was walking well without falling, but now was falling frequently. Id. Her mother denied other developmental regression, fever, or vomiting. Id. S.D. appeared well on examination, with normal ear and musculoskeletal examinations. Id. at 37-38. She was diagnosed with clumsiness that was possibly due an inner ear infection. Id. at 38. S.D. received a referral to an ear, nose, and throat ("ENT") specialist, and the pediatrician indicated that if that examination was normal, S.D. would need a magnetic resonance imaging ("MRI"). Id.

Later that same evening, S.D. was brought to the emergency room ("ER") at Johns Hopkins Hospital for clumsiness and emesis. Pet. Ex. 2 at 8. Petitioner reported that S.D.'s symptoms began about a week earlier, when she noticed that S.D. was "more fussy and clingy than usual" and "was falling more often." Id. She reported that one day earlier, S.D. was swaying and falling when trying to walk, losing balance easily. Id. S.D. had thrown up two bottles of milk a few days earlier and had been throwing up in her crib. Id. S.D. was also sleeping more than usual over the past week. Id. Petitioner denied recent injury or fever. Id. S.D. had been meeting developmental milestones. Id. On examination, S.D. was afebrile and alert, but fussy. Id. at 9. She had a normal neurologic examination except for a wide-based gait, losing balance, and falling. Id. She grabbed well with her hands. Id. Lab tests revealed an elevated lactate dehydrogenase level ("LDH") of 457. Id. S.D. was admitted for observation. Id.

The admission note stated that S.D. presented with five to seven days of vomiting, fatigue, and clumsiness. Pet. Ex. 2 at 13. "For the past [seven] days, [Petitioner] ha[d] noticed that [S.D.] ha[d] been fussy, clingy, and not acting like her cheerful self." Id. S.D. was also sleeping much more than usual and having increased falls. Id. Petitioner stated that S.D. had always had a wide-based gait, and she had not observed any changes. Id. Petitioner also reported that S.D. said "dada" and maybe a few words, but she was not sure. Id. at 14. S.D. was "[f]riendly, social, interactive[,] and cheerful at baseline." Id. Notable labs included elevated white blood cell count ("WBC") at 14, calcium level at 11, elevated LDH, and an elevated platelet count. Id. at 16. The differential diagnoses included a brain tumor and post-viral cerebellitis, and "GPS" given receipt of vaccinations two weeks earlier. Id. at 18. However, it was noted that S.D.'s examination was not indicative of progressive weakness, and her neurological findings had not worsened during her hospitalization. Id.

On May 19, 2016, S.D. was seen by neurology. Pet. Ex. 2 at 19-22. Her history noted two weeks of a "wider-based, unsteady gait . . . describe[d] as 'wobbly,'" with increased falling. Id. at 20. Her examination was normal with down-going toes and normal reflexes, and she was

able to "[r]each[] for objects without ataxia." Id. at 21-22. The only abnormalities were S.D.'s wide-based gait with unsteadiness and falling backwards at times with her arms raised for balance. Id. The differential diagnoses were brain tumor, acute cerebellar ataxia, and meningitis/encephalitis. Id. at 22. An MRI and lumbar puncture were recommended and several viral studies were ordered. Id. It was noted that S.D. had an elevated LDH and thrombocytosis (elevated platelet count) that was consistent with a viral etiology. Id. at 161.

A brain MRI was normal. Pet. Ex. 2 at 30. S.D. had normal/negative urine toxicology screen, ammonia level, repeat lactate level, and decreasing LDH (457 to 382), as well as a slightly elevated WBC at 14.82 with atypical lymphocytes possibly suggestive of a viral process. Id. at 31. Cerebrospinal fluid ("CSF") analysis showed a glucose level of 63, protein level of 38, WBC of 71, and red blood cell count ("RBC") of 44000. Id. at 32. CSF viral PCRs were negative for herpes simplex virus ("HSV"), cytomegalovirus ("CMV"), Epstein-Barr virus ("EBV"), varicella zoster virus ("VZV"), and enterovirus. Id. at 32, 40, 49-51, 53. A comprehensive metabolic panel was normal except for elevated potassium, calcium, alanine aminotransferase ("ALT"), and anion gap and low creatinine. Id. at 44. Neurology did not endorse a bacterial process following the lumbar puncture results. Id. at 172. They found the lumbar puncture was "possibly consistent with a viral process." Id. at 177-78. S.D.'s providers did not identify a definitive cause of S.D.'s condition. Id.

S.D. was eating, drinking, and feeling better, so she was discharged on May 21, 2016. Pet. Ex. 2 at 181-82. That day, her examination was normal except for a slightly wide and unsteady gait. Id. at 181. The discharge summary indicated that the etiology of S.D.'s symptoms "remain[ed] uncertain" but "appear[ed] possibly consistent with viral process." Id. at 29, 182. A neurology consultation on day of discharge indicated that no truncal ataxia was observed while S.D. was standing in the crib or moving. Id. at 111. S.D. was able to bounce up and down while holding onto a handrail. Id.

S.D. saw her pediatrician on May 23, 2016 for post-hospitalization follow-up. Pet. Ex. 1 at 39. Assessment was acute cerebellar ataxia. Id. at 40. She returned to pediatrics on May 27, with non-bilious vomiting, decreased appetite, and lethargy for one day. Id. at 41. S.D. did not have other symptoms, but the provider wanted to rule out intracranial pressure as the cause of S.D.'s vomiting, so S.D. was sent to the ER. Id. at 42. S.D. was brought to the Johns Hopkins ER where her examination revealed no gait disturbance and no ataxia. Pet. Ex. 2 at 75. The examination was normal with no gross neurologic deficits noted. Id. at 73. The diagnosis was constipation, and she was discharged to home. Id. at 74.

On July 21, 2016, S.D. had a pediatric visit for lethargy and tugging at her ear. Pet. Ex. 1 at 43. It was noted that she was walking well but preferred to lie down. Id. The assessment was lethargy. Id. Observation was recommended as she had no other issues. Id.

On July 27, 2016, S.D. saw neurologist, Yuval Shafrir, M.D. Pet. Ex. 3 at 70. The initial history noted that S.D. was the result of a high-risk pregnancy, induced at 37 weeks with a birth weight of five pounds. Id. She walked at eight months and said "dada" at seven months but then stopped. Id. at 71. He further noted that S.D.'s older brother had significant developmental problems; he was an early walker but late talker, and had sensory problems similar to S.D. Id.

Dr. Shafrir reviewed S.D.'s hospitalization records and stated the MRI was read as normal. Id. at 73. However, Dr. Shafrir was worried that there were "signal changes on FLAIR in the subcortical areas bilaterally" and "quite significant signal change in the left peritrigonal area." Id. Although several providers said that S.D. was interactive, Dr. Shafrir noted that S.D.'s mother now recalled that at the time of her admission she acted "completely oblivious to her surroundings." Id. S.D.'s mother reported that after her hospitalization, S.D. ignored other children, preferred to be alone, flapped her hands, and had sensory issues. Id. at 74. S.D.'s mother also reported excessive sleepiness. Id. S.D.'s mother reported that although S.D. was walking better compared to her condition at the time of hospital discharge, she was still quite unsteady. Id.

On examination, S.D. "was completely unresponsive to her name," "did not follow [a] pointing finger," and "did not make eye contact" when being examined. Pet. Ex. 3 at 74. She also did not respond to her mother (Petitioner) calling her. Id. Dr. Shafrir's impression was that S.D. developed an "encephalitic process [] with acute ataxia, sleepiness[,] and vomiting with decrease[d] . . . mental status (according to mother's description)." Id. at 75. S.D. "continued, however, in a full-blown autistic syndrome." Id. Her gait improved but her sleepiness had not, suggesting a hypothalamic or brainstem injury. Id. He recommended repeat MRI, lumbar puncture, and blood work, in addition to a video electroencephalogram ("EEG"). Id. He also suggested a milk-free diet and hearing test, and requested S.D.'s immunization record. Id. at 76. He also considered intravenous immunoglobulin ("IVIG") and steroids. Id.

Repeat brain MRI on August 3, 2016, interpreted by Dr. David Moss, showed "[s]everal small foci of subtle FLAIR signal mostly in the subcortical parieto-occipital white matter nonspecific but certainly consistent with sequelae of encephalitis." Pet. Ex. 15 at 90. S.D. had a 24-hour video EEG on August 4, 2016, which indicated bilateral cortical dysfunction that was maximal in the right posterior quadrant and left temporal region. Pet. Ex. 3 at 47-48.

On August 23, 2016, S.D. had a developmental assessment by the Maryland Infants and Toddlers Program. Pet. Ex. 7 at 20-24. She had age-appropriate gross motor and self-help skills. Id. at 21. She had delayed cognition, fine motor, and social emotional skills. Id. at 22. At the time of her assessment, she had difficulty responding to her name, attaching meaning to object labels, engaging others, and maintaining attention to communication and sound awareness. Id. at 23.

On August 29, 2016, S.D. returned to Dr. Shafrir. Pet. Ex. 3 at 41. Dr. Shafrir noted that after the previous visit, S.D. underwent several studies. Id. She had an abnormal 24-hour video EEG that indicated the presence of bilateral cortical dysfunction. Id. A complete blood count ("CBC"), complete metabolic panel, chromosomal microarray, and vitamin B12 and folic acid levels were all normal. Id. Immunoglobulins, total carnitine, and free carnitine were also normal. Id. A celiac antibody panel was negative, as was a lumbar puncture. Id. at 42. Mumps and measles antibodies were positive. Id. at 41. CSF and multiple sclerosis ("MS") profile did not show any intrathecal production of Immunoglobulin ("Ig") G, oligoclonal bands were negative, and varicella virus DNA in CSF was negative, as was a viral culture. Id. at 42. Rubella RNA PCR testing was negative. Id. There was no evidence of folate receptor antibody syndrome, and an autoimmune encephalopathy panel on CSF and serum was negative. Id. At

this visit, Dr. Shafrir also documented S.D.'s vaccine history and noted that "the possibility that acute cerebellar ataxia was a response to the vaccination[] is very likely." Id. Dr. Shafrir observed that there was some improvement in S.D.'s sleepiness since he last saw her. Id. S.D.'s father stated that S.D.'s balance was back to normal and that she was running in the office. Id. However, her interaction was still abnormal. Id. Dr. Shafrir discussed S.D.'s autism presentation and noted that he had never seen or heard about a similar case, "although cerebellar abnormalities associate[ed] with autism were extensively reported in the literature." Id. at 44. He recommended starting IVIG and steroids. Id.

On September 15, 2016, S.D. began four days of IVIG treatments. Pet. Ex. 4 at 7. S.D. returned to Dr. Shafrir on September 26, 2016. Pet. Ex. 3 at 38. He noted that S.D. had been admitted for IVIG and then received a tapering dose of prednisone to prevent allergic meningitis sometimes associated with IVIG treatment. Id. Per her parents, S.D. appeared happier but there was no improvement in her core autistic symptoms. Id.

On October 7, 2016, S.D. was seen in the ER at Sinai Hospital of Baltimore. Pet. Ex. 4 at 240. She was admitted per Dr. Shafrir for a 24-hour EEG. Id. at 242. The admission note stated that S.D. had a history of "MMR acute cerebellar ataxia [status post] IVIG now with [one] week of staring spells worsening over the past [three] days." Id. at 240. The spells lasted less than five minutes and then she often covered her eyes and fell asleep. Id. On examination, there were no focal neurologic deficits. Id. at 241. She was admitted for concerns of absence seizures and to undergo an EEG and labs. Id. at 241-42. Diagnoses included cerebellar ataxia, developmental ataxia, and possible autoimmune encephalopathy. Id. at 232. Petitioner reported that S.D. had regained most motor deficits and was still working on improving fine coordination. Id. at 257. However, her language deficits had not improved and there was also concern for autism. Id. at 257-58. S.D. underwent the 24-hour EEG and was discharged home. Id. at 260. Dr. Shafrir interpreted the EEG as showing the presence of bitemporal cortical dysfunction. Pet. Ex. 3 at 36. He noted that the "[t]he findings [were] quite unusual and nonspecific." Id.

S.D. saw Dr. Shafrir in follow-up on October 20, 2016. Pet. Ex. 3 at 27. He reviewed the Maryland Infants and Toddlers Program assessment. Id. He stated that he was familiar with literature on autoimmune encephalopathies, acute cerebellar ataxia, and vaccination reactions, but was "not familiar with any case like [S.D.'s]." Id. at 29-30. Dr. Shafrir reported that he was pleased with her response to IVIG, although he did not specify any improvement. Id. at 30. He prescribed IV steroids followed by taper doses. Id.

S.D. followed up with Dr. Shafrir on November 14, 2016. Pet. Ex. 3 at 24. He described S.D. as having a "very unusual neurological condition, starting with acute cerebellar ataxia following MMR vaccination and continued with excessive sleepiness and then appearance of full-blown autistic symptoms." Id. Dr. Shafrir noted that S.D. had progressive diffuse slowing on her EEG and minimal MRI changes. Id. He stated that S.D. had IVIG treatments that produced some improvement but not a dramatic change in her autistic symptoms. Id. at 24-25.

A video EEG from June 23, 2017 was abnormal due to "intermittent high amplitude rhythmic slowing in the left than the right posterior quadrant, spreading to the left and the right temporal region." Pet. Ex. 3 at 23. The same day, Dr. Shafrir sent a note to S.D.'s pediatrician

reporting an "autistic regression, which followed acute cerebellar ataxia, probably induced by the MMR vaccination. She had significant encephalopathy following her recovery from acute cerebellar ataxia with excessive sleepiness and slowing on her EEG." Id. at 5. He noted that S.D.'s receptive language was very poor, and that she may cover her eyes but only occasionally covered her ears and was rarely involved in visual self-stimulation. Id. at 6. She walked on tiptoes and occasionally flapped her hands. Id. She was curious. Id. She had a "special educator," who saw her once or twice a month, and a speech therapist. Id. She went to a "center" four times a week where she "attend[ed] a classroom with [three] other children." Id. She was playing well in the office but ignored verbal stimuli and her name being called, and rarely followed a pointed finger. Id. An examination, including her gait, was normal. Id. at 6-7. Dr. Shafrir recommended continuing IVIG. Id. at 8.

S.D. was seen in the ER on June 27, 2017 for a prolonged staring spell lasting about 10 minutes, during which she was not responsive. Pet. Ex. 4 at 382. No abnormal movements were noted. Id. After the episode resolved, she went back to being herself. Id. The etiology of her staring spells was unknown, but they could have been absence seizures. Id. at 386. Dr. Shafrir noted that S.D. was "demonstrating signs and symptoms clearly of autism spectrum disorder without much improvement in communication despite what seems like extensive treatment and will give IVIG for possible ataxia." Id.

On August 7, 2017, S.D. had her two-year well-child examination. Pet. Ex. 1 at 53. The pediatrician noted that S.D. had been diagnosed with autism, that she had a "[r]eaction to MMR," and that she was on IVIG therapy. Id. An examination was normal. Id. S.D.'s grandmother, who brought her in, refused vaccines. Id. at 54.

In a note to S.D.'s pediatrician dated December 21, 2017, Dr. Shafrir reported that S.D. was given a second round of IVIG on June 26, 2017 and had a more dramatic response than she had after the first round of treatment. Pet. Ex. 3 at 9. After seeing S.D. for follow-up in July 2017 after IVIG, he wanted her to repeat the IVIG in six to eight weeks, but her parents did not bring her in as planned. Id. Dr. Shafrir noted that S.D. had lost all the words she had developed and had stopped babbling, which she was doing well until a month earlier. Id. She also stopped making eye contact with strangers and had some new sensory symptoms of covering of her ears and eyes. Id. However, she was not irritable. Id. She had episodes of unexplained laughter and staring spells. Id. Dr. Shafrir recommended observation at this point and IVIG if she developed worsening symptoms. Id. at 11.

On April 19, 2018, S.D. saw Dr. Shafrir for follow-up, during which her parents reported an "abrupt change in her behavior." Pet. Ex. 6 at 12-13. They reported that she had a better response to her name, albeit inconsistently, and that she was making more sounds. Id. at 13. However, she began to have episodes of frequent hitting and was hitting objects with her head. Id. She also had episodes of laughter without obvious triggers, increasing in the last week. Id. Dr. Shafrir noted that he had not seen S.D. for three months, although he had advised her parents that she needed to return two weeks after the last visit. Id. at 12-13. S.D.'s parents had kept her on "a medium dose" of prednisone, rather than weaning it as instructed. Id. at 13.

On April 21, 2018, Dr. Shafrir admitted S.D. to the hospital for alleged worsening autoimmune encephalitis. Pet. Ex. 6 at 23. He noted that since S.D.'s office visit two days earlier, her symptoms "significantly worsened;" she was not making eye contact and she had "some bizarre movement and activities." Id. She continued to have uncontrollable laughing spells, she would bang her head, and she did not sleep most of the previous night. Id. at 24.

At the ER, S.D. did not exhibit these behaviors; she was nonverbal, very active, and was toe walking, but in no distress. Pet. Ex. 10 at 23. An admission note stated that just after S.D. turned one, she began to show regression in her milestones and experienced staring spells. Id. at 43. She had multiple EEGs and was treated with IVIG for presumed autoimmune encephalitis. Id. Over the last two years, her IVIG was given every eight weeks and, more recently, about every 12 weeks. Id. She received steroids after her IVIG courses. Id. She had a week of self-injurious behaviors including banging her head against the walls as well as laughing spells. Id. at 44. Assessment was "autism, autoimmune encephalitis[,] and a questionable seizure history." Id.

A 24-hour video EEG from April 22, 2018 was "mildly abnormal" due to "intermittent rhythmic slowing in the right posterior quadrant of the bitemporal area." Pet. Ex. 6 at 17-19. This was a nonspecific finding that was thought to possibly represent some degree of cortical dysfunction in that region. Id. at 19.

On October 7, 2021, at age six years and five months, S.D. established treatment with a new neurologist at the Kennedy Krieger Institute, Christina Morris-Berry, M.D. Pet. Ex. 33 at 13. Dr. Morris-Berry reviewed S.D.'s prior records and stated that S.D. was admitted to Johns Hopkins Hospital for two days in May 2018, where S.D.'s MRI and lumbar puncture were unremarkable. Id. at 14. Dr. Morris-Berry further noted S.D.'s treatment with Dr. Shafrir, who diagnosed encephalitis with autism. Id. Dr. Morris-Berry documented that S.D.'s parents "have not vaccinated again because of Dr. Shafrir" and she "strongly recommend[ed] resuming vaccinations on a catch-up schedule with pediatrician at this point, as the benefit of vaccinations far outweigh risk." Id. at 16, 18. The assessment stated that S.D. had been free of staring spells for two years, was doing well, and had no new developmental regressions. Id. at 18.

2. Petitioner's Affidavit

Petitioner stated that prior to S.D.'s MMR vaccination on April 27, 2016, S.D. was generally healthy with no significant medical history. Pet. Ex. 5 at ¶¶ 4-5. Petitioner first noticed an issue with S.D. when she "kept falling backwards and couldn't find her balance when walking." Id. at ¶ 6. Petitioner thought these issues were due to her "awkward gait and teething" because she "heard . . . the pain from teething can mess with a child's balance." Id. However, when S.D. did not improve, she took her to the ER at Johns Hopkins on May 18, 2016. Id. at ¶¶ 6-7. "The doctors [at Johns Hopkins] were very concerned, but couldn't provide clear answers other than that [S.D.'s] brain was inflamed." Id. at ¶ 7. Petitioner explained S.D. "went from achieving and exceeding all developmental milestones to wanting to sleep all day every day" and would not "babble, speak, or make eye contact." Id. at ¶ 8.

III. STANDARDS FOR ADJUDICATION

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). To receive compensation through the Program, Petitioner must prove either (1) that S.D. suffered a "Table injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that S.D. suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). There is a statutorily prescribed presumption of causation for a Table injury. § 14(a). A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). "[F]actors unrelated to the administration of the vaccine" do not include "any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness, or condition." § 13(a)(2)(A).

Relevant here is the Qualification and Aids to Interpretation ("QAI") for a Table claim of encephalitis:

> A vaccine recipient shall be considered to have suffered encephalitis if an injury meeting the description below of acute encephalitis occurs within the applicable time period and results in a chronic encephalopathy[5] . . . .
>
> (i) Acute encephalitis. Encephalitis is indicated by evidence of neurologic dysfunction, as described [below], plus evidence of an inflammatory process in the brain, as described [below].
>
> (A) Evidence of neurologic dysfunction consists of []:
>
> (1) One of the following neurologic findings referable to the [central nervous system ("CNS")]: Focal cortical signs (such as aphasia, alexia, agraphia, cortical blindness); cranial nerve abnormalities; visual field defects; abnormal presence of primitive reflexes (such as Babinski's sign or sucking reflex); or cerebellar dysfunction (such as ataxia, dysmetria, or nystagmus) . . . .

[5] According to the Vaccine Injury Table,"[a] chronic encephalopathy occurs when a change in mental or neurologic status, first manifested during the applicable Table time period as an acute encephalopathy or encephalitis, persists for at least [six] months from the first symptom or manifestation of onset . . . of an acute encephalopathy or encephalitis." 42 C.F.R. § 100.3(d).

> (B) Evidence of an inflammatory process in the brain ([CNS] or CNS inflammation) must include [CSF] pleocytosis (> 5 [WBC]/mm^3 in children > 2 months of age and adults; > 15 WBC/mm3 in children < 2 months of age); or at least two of the following:
> (1) Fever (temperature ≥ 100.4° Fahrenheit);
> (2) [EEG] findings consistent with encephalitis, such as diffuse or multifocal nonspecific background slowing and periodic discharges; or
> (3) Neuroimaging findings consistent with encephalitis, which include, but are not limited to brain/spine [MRI] displaying diffuse or multifocal areas of hyperintense signal on T2–weighted, diffusion-weighted image, or fluid-attenuation inversion recovery sequences.

42 C.F.R. § 100.3(c)(3)(i). Exclusionary criteria for an encephalitis claim indicates that

> [r]egardless of whether or not the specific cause of the underlying condition, systemic disease, or acute event (including an infectious organism) is known, encephalitis shall not be considered to be a condition set forth in the Table if it is shown that the encephalitis was caused by:
> (A) An underlying malignancy that led to a paraneoplastic encephalitis;
> (B) An infectious disease associated with encephalitis, including a bacterial, parasitic, fungal or viral illness (such as herpes viruses, adenovirus, enterovirus, West Nile Virus, or human immunodeficiency virus), which may be demonstrated by clinical signs and symptoms and need not be confirmed by culture or serologic testing; or
> (C) Acute disseminated encephalomyelitis (ADEM). Although early ADEM may have laboratory and clinical characteristics similar to acute encephalitis, findings on MRI are distinct with ADEM displaying evidence of acute demyelination (scattered, focal, or multifocal areas of inflammation and demyelination within cerebral subcortical and deep cortical white matter; gray matter involvement may also be seen but is a minor component); or
> (D) Other conditions or abnormalities that would explain the vaccine recipient's symptoms.

Id. at § 100.3(c)(3)(ii). For a Table injury of encephalitis post-MMR vaccination, "the time period in which the first symptom or manifestation of onset . . . is to occur" is five to 15 days (not less than five days and not more than 15 days). Id. at § 100.3(a)(III)(B).

In reviewing the evidence, medical records, specifically contemporaneous medical records, are presumed to be accurate and generally "warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of

Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11–685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

Further, a petitioner must show that they have suffered the residual effects or complications of the alleged injury for more than six months after administration of the vaccination at issue. § 11(c)(1)(D)(i).

IV. EXPERT OPINIONS AND CAUSATION ANALYSIS

Petitioner here has alleged S.D. suffered a Table injury of encephalitis as a result of an MMR vaccination administered on April 27, 2016. The parties dispute the diagnosis of S.D.'s injury and whether the criteria have been met for a Table claim of encephalitis. Joint Submission, filed Sept. 1, 2023, at 1 (ECF No. 96);[6] Pet. Mot. at 1; Resp. Response at 13-16. Further, Respondent asserts that S.D. has not suffered the residual effects of her condition for more than six months after administration of the vaccine. Resp. Response at 20-21.

A. Experts Reports[7]

1. Petitioner's Expert, Dr. Frederick Nahm, M.D., Ph.D.

a. Qualifications

Dr. Nahm is a practicing neurologist with board certifications in neurology and electrodiagnostic medicine and over 20 years of clinical experience. Pet. Ex. 16 at 1; Pet. Ex. 17 at 3. Dr. Nahm received a Ph.D. in neuroscience from the University of California, San Diego in 1994 and his M.D. from the University of Michigan Medical School in 1996. Pet. Ex. 17 at 1.

[6] The party's joint submission does not clearly reflect a Table claim. See Joint Submission, filed Sept. 1, 2023 (ECF No. 96). However, Petitioner alleged a Table claim in her Petition and in her motion for a ruling on the record. See Petition at 1; Pet. Mot. at 1. Both party's experts provided opinions about a Table claim as well as a causation-in-fact claim. Moreover, Respondent's brief addresses a Table claim. See Resp. Br. at 13-20. Because the undersigned finds that Petitioner has proven a Table claim for encephalitis following an MMR vaccination, the causation-in-fact claim is not analyzed.

[7] For reasons explained in the footnote above, the undersigned does not discuss the experts' opinions related to a causation-in-fact claim of encephalitis.

Following medical school, he completed a neurology residency, ethics fellowship, clinical neurophysiology fellowship, and neuromuscular fellowship. Id. at 2. He has held teaching appointments at Yale University, Harvard Medical School, and University of California, San Diego. Pet. Ex. 16 at 1. Dr. Nahm founded a private neurology practice in 2002 and continues to be actively involved in clinical neurology. Id.; Pet. Ex. 17 at 1.

b. Opinions

i. Initial Report

Dr. Nahm correctly noted that "encephalitis secondary to MMR is a Table injury if occurring between 5-15 days of vaccination." Pet. Ex. 16 at 17.

Beginning with his opinion regarding onset, Dr. Nahm opined that S.D. had a neurological injury caused by the MMR vaccine, which began eight to 14 days after vaccination. Pet. Ex. 16 at 2. He opined that prior to vaccination, S.D. was neurologically intact, with no history of gait disorder. Id. at 18. On the day she received her vaccination, S.D. met most of her developmental milestones; she was walking, pulling to stand, playing peek-a-boo, and able to thumb finger grasp. Id. Based on the pediatric records dated May 18, 2016, Dr. Nahm opined that S.D. developed gait instability and increased falls, clumsiness, and sleepiness approximately May 11, 14 days after receiving the MMR vaccination. Id. (citing Pet. Ex. 1 at 37). And according to the records of the initial neurology consult, S.D.'s symptoms began around May 5, about eight days post-vaccination. Id. (citing Pet. Ex. 2 at 19). Thus, Dr. Nahm opined that the range of onset is eight to 14 days after MMR vaccination. Id.

Dr. Nahm further noted that S.D. was diagnosed with "acute cerebellar ataxia, which is clinical evidence for neurological dysfunction. This clinical diagnosis of cerebellar ataxia 'stands in' for a diagnosis of encephalitis when taken together with the totality of her clinical history . . . ." Pet. Ex. 16 at 18-19. He cited a paper by Plesner et al.,[8] reporting on a study done in Denmark about adverse reports of gait disturbance in children after the MMR vaccination. Pet. Ex. 24 at 2. The authors documented that cerebellar ataxia has been described after the measles vaccine and that it was also a well-known symptom of encephalitis following natural infections with measles, mumps, and rubella virus. Id. at 6.

In addition to having neurological dysfunction due to her diagnosis of cerebellar ataxia, Dr. Nahm explained that S.D. also had evidence of an inflammatory process in the brain. Pet. Ex. 16 at 19. He noted CSF studies revealed elevated WBC, which is "entirely consistent with encephalitis." Id. Although the lumbar puncture was traumatic due to the presence of a significant number of RBC in the CSF fluid, Dr. Nahm applied a correction factor to determine whether or not there was CSF pleocytosis, or an increase in cells, to accurately assess the

[8] A-M Plesner et al., Gait Disturbance Interpreted as Cerebellar Ataxia After MMR Vaccination at 15 Months of Age: A Follow-up study, 89 Acta Paediatrica 58 (2000).

presence of inflammation. Id. Using a correction factor from a published study by Lyons et al.,[9] Dr. Nahm determined that S.D.'s corrected CSF WBC would be 13.68 (tube #1) as compared with the actual value of 20, and 50 (tube #4) as compared with the actual value of 71. Id. (citing Pet. Ex. 22 at 2). According to Dr. Nahm, S.D. "had an elevated CSF WBC that cannot be accounted for by the traumatic lumbar puncture." Id.

Lyons et al. conducted a retrospective analysis of lumbar punctures from 20 participating centers and reviewed 20,319 lumbar punctures of which 2,880 were traumatic. Pet. Ex. 22 at 2. CSF pleocytosis was defined as a WBC equal to or greater than 10 cells/mm^3 for infants 29-60 days of age. Id. Using a linear regression analysis, the authors derived a CSF WBC correction factor. Id. The primary goal of the study was "[t]o determine the optimal correction factor for [CSF] [WBC] counts in infants with traumatic lumbar punctures" to better diagnosis bacterial meningitis in infants (although the correction factor was not limited to that clinical scenario). Id. Application of the applied correction factor "substantially reduced" the number of infants diagnosed with CSF pleocytosis. Id. The authors emphasized that assessing CSF WBC must be considered "in the context of the infant's other clinical and laboratory factors." Id. at 7.

Based on the Table QAI, and the application of the Lyons et al. correction factor, Dr. Nahm opined that S.D.'s corrected WBC values would be greater than 5 cells/mm3 (the value for children over the age of two months), evidencing "an inflammatory process in the brain." Pet. Ex. 16 at 19. He further opined that "[w]hen taken together with her evidence of neurological dysfunction (cerebellar ataxia), both provide the proof needed to demonstrate encephalitis." Id.

According to Dr. Nahm, further evidence of inflammation in the brain is found in the October 7, 2016 abnormal EEG, which showed "very high amplitude rhythmic bitemporal slow theta activity indicative of cortical dysfunction." Pet. Ex. 16 at 20 (citing Pet. Ex. 3 at 36). S.D. also had an abnormal EEG that showed "bitemporal slowing" in June 2017. Id. (citing Pet. Ex. 3 at 23). Dr. Nahm opined that both of these EEGs show persistent post-encephalitic brain injury. Id. Dr. Nahm also discussed S.D.'s abnormal brain MRI on August 3, 2016. Id. It showed "several small foci of subtle FLAIR signal mostly in the subcortical parieto-occipital white matter nonspecific but consistent with sequelae of encephalitis." Id. (citing Pet. Ex. 15 at 90). Dr. Nahm concluded that both the abnormal EEGs and MRI "suggest persisting neurological dysfunction/injury as a result of [S.D.'s] encephalitis." Id. As additional evidence of an inflammatory process in the brain, Dr. Nahm noted that S.D. had a "dramatic response to IVIG treatment" in that her cerebellar ataxia improved, pointing to "an underlying immunological process." Id.

Next, Dr. Nahm turned to the issue of exclusionary criteria for a Table claim of encephalitis after MMR vaccination. Pet. Ex. 16 at 20. He opined that S.D. "had no exclusion criteria; there was no documentation of underlying malignancy, infectious disease, diagnosis of ADEM, or other abnormalities that would explain [S.D.'s] symptoms.: Id. He noted "[a]ll [] tests for a viral antigen as a cause of [S.D.'s] encephalitis were negative" and "[a]dditional

[9] Todd W. Lyons et al., Interpretation of Cerebrospinal Fluid White Blood Cell Counts in Young Infants with a Traumatic Lumbar puncture, 69 Annals Emergency Med. 622 (2017).

studies did not show any chromosomal abnormalities, inborn errors of metabolism, tumor, or other diseases that would otherwise explain [S.D.'s] clinical course." Id.

Regarding the six-month severity requirement, Dr. Nahm opined that S.D.'s "abnormal EEG on October 7, 2016 showing bitemporal cortical dysfunction[] indicates a persisting post-encephalitic condition [six] months from her MMR vaccination, which was still evident on the EEG on June 26/27, 2017, 13 months post MMR vaccination." Pet. Ex. 16 at 2.

ii. Supplemental Report

In his supplemental report, Dr. Nahm addressed Dr. Wiznitzer's contention that a URI was the more likely cause of S.D.'s acute cerebellar ataxia. Pet. Ex. 38 at 1-2. Dr. Nahm reiterated that the more common viral causes of this condition include those viruses that were tested for in S.D. and the results were all negative. Id. at 2. Therefore, Dr. Nahm concluded that "there is no objective evidence for any significant URI that would otherwise explain S.D.'s post-vaccine neurological condition." Id.

In response to Dr. Wiznitzer's opinion that S.D.'s August 2016 MRI "might be related to her IUGR status," Dr. Nahm explained that if the abnormalities seen on S.D.'s second MRI were caused by her IUGR, such abnormality would have also been seen on her initial MRI in May 2016. Pet. Ex. 38 at 2 (citing Resp. Ex. A at 13). Dr. Nahm stated "[t]here is no explanation offered as to how a chronic condition of IUGR could cause the brain MRI to go from normal to abnormal." Id. He also noted the treating radiologist interpreted the study to be consistent with the sequela of encephalitis. Id. (citing Pet. Ex. 15 at 90).

As for the experts' disagreement as to the significance of the elevated WBC in the CSF, Dr. Nahm reiterated the process used by Lyons et al. to correct for blood in the CSF. Pet. Ex. 38 at 3. He again opined that that S.D. had an elevated WBC even if one accounts for the blood in the CSF caused by the trauma of the procedure. Id.

Next, Dr. Nahm addressed Dr. Wiznitzer's opinion that the EEG abnormalities were caused by drowsiness or reflected an underlying autistic condition. Pet. Ex. 38 at 3-4. Dr. Nahm stated that it is misleading to suggest that the EEG showed slowing due to drowsiness. Id. Dr. Shafrir wrote, "The study indicates presence of bilateral cortical dysfunction, maximal in the right posterior quadrant and left temporal region. The similarity of the rhythmic slowing seen throughout the awake records, to the drowsy activity may suggest that it represents the mechanisms causing excessive sleepiness in the patient." Id. at 3 (quoting Pet. Ex. 3 at 41). Dr. Nahm explained that this interpretation means that "the EEG changes are what is causing the patient's excessive sleepiness" and not that "the EEG was abnormal because S.D. was drowsy." Id.

Dr. Nahm also took issue with Dr. Wiznitzer's opinion that S.D.'s abnormal EEG may have been caused by autism spectrum disorder. Pet. Ex. 38 at 3-4. He opined that "[t]o claim that the substantial EEG changes can be explained by a possible underlying condition of [autism spectrum disorder] is a stretch, if not mere conjecture." Id. at 4.

He further explained that "[w]hether S.D. ha[s] speech/language delays suggestive of [autism spectrum disorder] that predated her vaccinations on [April 27, 2016] is unclear as well as irrelevant, as no claim was made that her claimed [autism spectrum disorder] is vaccine related." Pet. Ex. 38 at 1. He found "[t]he relevant neurological changes post-vaccination were her impaired motor, balance, and behavior functions. Any form of pre-vaccine developmental delay experienced by S.D. does not alter a review of the medical records which clearly shows a discrete, acute, neurological syndrome which occurred only after her vaccinations." Id. And "regardless of any prior developmental delay, S.D. experienced a new, acute, and different neurological condition which was wholly distinct from any pre-vaccine developmental delay." Id.

In conclusion, Dr. Nahm opined that "S.D. developed an acute neurological condition with cerebellar ataxia, behavior changes, with documented abnormalities [on] MRI and EEG studies, and an abnormal WBC count in the CSF, all consistent with a post-vaccine encephalitis." Pet. Ex. 38 at 4.

2. Respondent's Expert, Dr. Max Wiznitzer[10]

a. Qualifications

Dr. Wiznitzer obtained his M.D. from Northwestern University in 1977. Resp. Ex. D at 1. He then completed a residency in pediatrics at Children's Hospital Medical Center in Cincinnati, Ohio; a fellowship at the Cincinnati Center for Developmental Disorders; a fellowship in pediatric neurology at the Children's Hospital of Philadelphia; and a fellowship in higher cortical functions at the Albert Einstein College of Medicine. Id. at 1-2. He is currently a pediatric neurologist at University Hospitals of Cleveland, as well as a professor of several subjects at Case Western University. Id. at 2; Resp. Ex. A at 1. He is board-certified in pediatrics and neurology, with a special qualification in child neurology, and neurodevelopmental disabilities. Resp. Ex. A at 1; Resp. Ex. D at 5. Dr. Wiznitzer's interests include autism spectrum disorders, developmental and behavioral disorders, and epilepsy in the neurodevelopmental disabilities population. Resp. Ex. A at 1. He "routinely read[s] [EEGs] and video EEGs" and treats patients with acute seizures and epilepsy. Id.

[10] The undersigned does not discuss the majority of Dr. Wiznitzer's opinions about autism spectrum disorder, as Petitioner does not allege that S.D.'s vaccination caused autism or an autism spectrum disorder. Therefore, these opinions are not relevant to the Table criteria for encephalitis. Further, there are no medical record references about autism or autism spectrum disorders by S.D.'s treating physicians prior to vaccination or during the period of time relevant to the neurological injury at issue. The fact that autism and/or autism spectrum disorder or behavior may be referenced in subsequent medical records when S.D. became older is not an automatic bar to recovery of a Table claim. Each case must be resolved based on the facts and circumstances relevant to that specific case.

b. Opinions

i. Initial Report

Dr. Wiznitzer's acknowledged that when S.D. was initially hospitalized after her MMR vaccination, she had acute cerebellar ataxia with an onset of approximately May 10, 2016. Resp. Ex. A at 12.

Regarding S.D.'s CSF, and the elevated WBC reported, Dr. Wiznitzer attributed it to the traumatic procedure and blood in the CSF. Resp. Ex. A at 12. Based on his explanation, if there were pleocytosis due to inflammation, the ratio of RBC in the CSF would be smaller than in the blood. Id. Since he argued that is not the case, he concluded that there was no CSF evidence of an inflammatory process in the brain. Id. Dr. Wiznitzer does not cite any medical literature supporting his analysis of the CSF results.

Although Dr. Wiznitzer agreed that S.D.'s brain MRI on August 3, 2016, was abnormal, he disagreed that it was consistent with encephalitis because S.D.'s clinical history did not show a "significant impairment of consciousness associated with her acute ataxia." Resp. Ex. A at 12-13. Dr. Wiznitzer did not address the clinical history on presentation where S.D. was described as being fussy and clingy, and having fatigue, irritability, and vomiting. See id.; see also Pet. Ex. 2 at 19. He also suggested that the abnormal MRI could reflect her prenatal period of IUGR. Id.

Similarly, Dr. Wiznitzer agreed that S.D. had abnormal EEG findings of intermittent bilateral slowing, but he opined that these abnormalities could have "reasonable explanations" other than encephalitis. Resp. Ex. A at 13. He suggested that the EEGs could reflect drowsiness or abnormalities seen in children with autistic spectrum disorder. Id.

Next, regarding the Table exclusionary criteria, Dr. Wiznitzer attributed S.D.'s cerebellar ataxia to a prior URI that he maintained occurred about three weeks prior to the onset of ataxia. Resp. Ex. A at 12. He also opined that S.D. had a speech delay on the date of her vaccination which was the onset of an autism spectrum disorder, evidencing a pre-existing condition.[11] Id.

As for whether S.D.'s symptoms lasted six months, Dr. Wiznitzer opined that S.D.'s ataxia resolved by July-August 2016. Resp. Ex. A at 12.

ii. Supplemental Report

In his supplemental report, Dr. Wiznitzer responded to the opinions in Dr. Nahm's supplemental report, reiterating many of his prior opinions. Dr. Wiznitzer opined that S.D. did not have any evidence of acute encephalopathy or hyper somnolence during her initial hospitalization; that her increased WBC in her CSF is a factor of the blood in the CSF, and does

[11]The medical literature cited by Dr. Wiznitzer provides that autism is rarely diagnosed in children before the age of three, although some signs may be present as early as 18 months. Resp. Ex. A, Tab 4 at 1-2 (Chris Plauché Johnson, Recognition of Autism Before Age 2 Years, 29 Pediatrics Rev. 86 (2008)).

not reflect inflammation; that S.D.'s repeat MRI was normal and not consistent with the sequelae of encephalitis; and that the EEG done in August 2016 does not support a claim for encephalitis. Resp. Ex. C at 3-6.

In conclusion, Dr. Wiznitzer opined that "S.D. had a change in balance consistent with the diagnosis of acute cerebellar ataxia after her MMR vaccination with no evidence of inflammatory CSF pleocytosis on a traumatic lumbar puncture on [May 19, 2016], no evidence of changes consistent with encephalitis on MRI studies in May and August 2016, and no EEG study during her May 2016 admission." Resp. Ex. C at 6. He opined that "[t]hese findings are not sufficient" to meet the Table criteria for encephalitis. Id. He also maintained that S.D.'s autism spectrum disorder was the neurological dysfunction that persisted greater than six months, and that it was not caused by vaccinations. Id.

B. Analysis

After fully reviewing the parties' filings, the undersigned concludes that Petitioner has met her burden of demonstrating that S.D. suffered a Table encephalitis injury within five to 15 days of receiving an MMR vaccination for the following reasons.

1. Onset

Starting with onset, encephalitis secondary to MMR is a Table injury if occurring between five and 15 days post-vaccination. S.D. received her MMR vaccination on April 27, 2016. On May 18, 2016, S.D.'s mother reported to the pediatrician that S.D. had been clumsy and falling frequently for about one week. Using seven days as a measure of one week, this statement by S.D.'s mother places onset on May 11, or 14 days post-vaccination.

That same day, May 18, S.D. was taken to the Johns Hopkins ER for complaints of clumsiness and vomiting. Again, her mother reported that S.D.'s symptoms (fussy, clingy, and falling often) began about a week earlier. This report places onset on May 11, or 14 days post-vaccination. The admission notes for this date stated S.D. had five to seven days of vomiting and clumsiness (prior to hospital admission). The note also stated, "[f]or the past [seven] days, [Petitioner] ha[d] noticed that [S.D.] ha[d] been fussy, clingy," and having "increased falls." Pet. Ex. 2 at 13.

S.D. was seen by a neurologist on May 19, 2016, and the history documented that S.D. had two weeks of unsteady gait and increased falls. Using this report, onset would have been May 5, which would have been 8 days post-vaccination.

In summary, S.D.'s mother reported an onset of one week at the first visit to the pediatrician and to the ER providers on May 18. One record references onset five to seven days prior to admission. And the history taken by the most relevant specialist, a neurologist, documents an onset of two weeks. Taking all of the earliest reports into account, Petitioner most often reported an onset of one week, or seven days, with a range of five days to two weeks. Based on the medical records, the onset most frequently reported by Petitioner was that S.D.'s

symptoms begin one week prior to her presentation on May 18. This places onset on May 11, or 14 days after vaccination.

Turning to the experts' opinions, Petitioner's expert, Dr. Nahm, opines that S.D. received the vaccine on April 27, 2016 and developed symptoms on May 11, eight to 14 days later. He relies on notes from the records. The first note is Petitioner's report that S.D.'s symptoms began one week after vaccination. He also cites the reference to onset two weeks before S.D.'s initial evaluation.

Respondent's expert, Dr. Wiznitzer, opines that S.D. "had an occurrence of acute cerebellar ataxia" with onset of May 10, 2016. Resp. Ex. A at 12.

Thus, the experts agree that S.D.'s onset of clumsiness and falling, which represented cerebellar ataxia, was on or about May 10 to 11. Both dates are within the range of five to 15 days after vaccination.

Based on the medical records and expert opinions, the undersigned finds that onset of S.D.'s cerebellar ataxia was May 10 or 11, 2016. Both days are within the range of five to 15 days identified on the Table for claims of encephalitis after MMR vaccination. Therefore, S.D. meets the Table criteria for onset of encephalitis after the MMR vaccination.

2. Neurological Dysfunction

The next criterion that must be satisfied to prevail on a Table claim for encephalitis is that Petitioner must show "evidence of neurologic dysfunction" in S.D. 42 C.F.R. § 100.3(c)(3)(i). "Evidence of neurologic dysfunction consists of . . . [f]ocal cortical signs (such as aphasia, alexia, agraphia, cortical blindness); cranial nerve abnormalities; visual field defects; abnormal presence of primitive reflexes (such as Babinski's sign or sucking reflex); or cerebellar dysfunction (such as ataxia, dysmetria, or nystagmus)." Id. at § 100.3(c)(3)(i)(A).

The medical records show that when S.D. presented to her pediatrician on May 18, her mother reported that she had been very clumsy and was falling frequently. Later that day, at Johns Hopkins ER, S.D. was fussy, clingy, and falling often. The next day, May 19, neurological examination revealed that S.D. had a wide-based unstable gait, walked with her arms raised, and had intermittent falls. Differential diagnoses included acute cerebellar ataxia and meningitis/encephalitis. On May 23 and 27, 2016, S.D. returned to her pediatrician for follow-up examinations and at both visits, S.D.'s recent hospitalization and diagnosis of "acute cerebellar ataxia" were documented. Pet. Ex. 1 at 39-40, 42.

Dr. Nahm opines that S.D. "had symptoms of cerebellar ataxia and hypersomnia indicative of neurological dysfunction." Pet. Ex. 16 at 2. Although Dr. Wiznitzer did not opine that the vaccinations were causal, he agrees that that S.D. had "acute cerebellar ataxia." Resp. Ex. A at 12-14 (noting S.D. "had an occurrence of acute cerebellar ataxia . . .with onset around [May 10, 2016]," referring to "[S.D.'s] acute cerebella ataxia . . . [two] weeks after MMR," acknowledging "she had ataxia (the reason for her diagnosis of acute cerebella ataxia)," and concluding "she had acute cerebellar ataxia").

Additionally, Plesner et al. states that cerebellar ataxia is well known in connection with encephalitis. Dr. Nahm's opinion on this point is more persuasive because it is consistent with the Vaccine Injury Table, the QAI, the medical records, and the medical literature.[12]

In conclusion, both experts agree that S.D. had acute cerebellar ataxia within five to 15 days of receipt of the MMR vaccination. Pursuant to the above Table criteria, evidence of neurological dysfunction can be established by cerebellar dysfunction such as ataxia. Therefore, the undersigned finds by preponderant evidence S.D. had evidence of neurological dysfunction because she had acute cerebellar ataxia within five to 15 days of her MMR vaccination.

3. Inflammatory Process in the Brain

In addition to evidence of neurological dysfunction, Petitioner must show "evidence of an inflammatory process in the brain" of S.D. for an encephalitis claim. 42 C.F.R. § 100.3(c)(3)(i).

> Evidence of an inflammatory process in the brain . . . must include [CSF] pleocytosis (> 5 [WBC]/mm^3 in children > 2 months of age and adults; . . . or at least two of the following:
> (1) Fever (temperature ≥ 100.4° Fahrenheit);
> (2) [EEG] findings consistent with encephalitis, such as diffuse or multifocal nonspecific background slowing and periodic discharges; or
> (3) Neuroimaging findings consistent with encephalitis, which include, but are not limited to brain/spine [MRI] displaying diffuse or multifocal areas of hyperintense signal on T2–weighted, diffusion-weighted image, or fluid-attenuation inversion recovery sequences.

Id. at § 100.3(c)(3)(i)(B).

S.D.'s CSF revealed elevated WBC of 20 (Tube #1) and 71 (Tube #2), which meet the criteria set forth above, however the fluid contained blood due to a traumatic procedure. Neither the Table nor the QAI address the question of what to do when there is an elevated WBC so as to meet the definition of pleocytosis, but the CSF also contains blood skewing the results. Dr. Nahm opined that even accounting for the increased red blood cells due to the traumatic procedure, the corrected results showed pleocytosis that meet and exceed the Table criteria of greater than five, consistent with an inflammatory process in the brain. Dr. Nahm relies on Lyons et al., a scholarly paper published in a peer reviewed journal, in support of his analysis, and specifically for the calculation he used to account for the elevated WBC attributable to the

[12] Of note, Dr. Wiznitzer states that "[S.D.] had acute cerebellar ataxia." Resp. Ex. A at 14. Thus, he acknowledges her diagnosis of cerebellar ataxia. Therefore, there does not appear to be any dispute between the experts that S.D. meets the Table criteria for "evidence of neurological dysfunction." 42 C.F.R. § 100.3(c)(3)(i). However, Dr. Wiznitzer also opines there is "no evidence of encephalitis." Resp. Ex. A at 12. The undersigned finds this aspect of Dr. Wiznitzer's opinion inconsistent with the Vaccine Injury Table QAI for "[a]cute encephalitis." 42 C.F.R. § 100.3(c)(3)(i).

traumatic lumbar puncture. Thus, even correcting for the traumatic lumbar puncture, Dr. Nahm opined that S.D. had elevated WBC in her CSF.

Dr. Wiznitzer disagrees, stating that because the lumbar puncture was traumatic, it resulted in elevated RBC that artificially elevated the CSF cell count, and thus, the increased cell count was not due to inflammation.

The CSF showed elevated WBC as required by the criteria. Although the results are artificially elevated due to the presence of blood, the undersigned finds Dr. Nahm's approach and opinions more persuasive because he cited reliable medical literature to explain his use of the correction factor to determine whether there was evidence of inflammation. Moreover, as Lyons et al. explains, the CSF results are to be viewed in the context of the clinical course. Here, the clinical course was consistent with cerebellar ataxia.

Moreover, there is preponderant evidence of CNS inflammation based on S.D.'s MRI and EEG tests. S.D.'s initial brain MRI was done on May 19, 2016, during her hospitalization, and it was interpreted as normal. However, when it was reviewed by Dr. Shafrir on July 27, 2016, he noted signal changes in the "subcortical areas bilaterally" and the "left peritrigonal area." Pet. Ex. 3 at 73. Dr. Shafrir's impression was that S.D. had an encephalitic process. Id. at 75. Additionally, the interpreting radiologist, Dr. Moss, found the repeat brain MRI done August 3, 2016 showed "[s]everal small foci of subtle FLAIR signal mostly in the subcortical parieto-occipital white matter nonspecific but certainly consistent with sequelae of encephalitis." Pet. Ex. 15 at 90. The undersigned finds that the opinion of the treating physicians, Dr. Shafrir and Dr. Moss, to be the most accurate and reliable as to the results of the MRI study.

The opinions of Petitioner's treating physicians are afforded substantial weight. Capizzano, 440 F.3d at 1319-20. Additionally, medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528.

Therefore, the undersigned finds that the MRI studies done in May and August 2016, showing the abnormalities described above, are "consistent with encephalitis" and evidence of CNS inflammation. 42 C.F.R. § 100.3(c)(3)(i)(B)(3).

Next, S.D.'s initial EEG done in August 2016 was abnormal, showing "very frequent intermittent rhythmic slowing seen mainly in the right posterior quadrant and the left temporal region." Pet. Ex. 3 at 47-48. Dr. Shafrir interpreted the study as showing "bilateral cortical dysfunction," and he noted that "rhythmic slowing [was] seen throughout the awake record." Id. at 48. A subsequent EEG done in June 2017 was also abnormal, again showing persisting bitemporal slowing. Dr. Nahm opined that "[t]hese studies indicate persisting post-encephalitic brain injury." Pet. Ex. 16 at 20. Dr. Wiznitzer did not refute the EEG interpretation of Dr. Nahm, specifically that the EEGs showed post-encephalitic brain injury, but he questioned whether there was a clinical correlation (was there "normal drowsiness or brain dysfunction causing the drowsiness") and he disputed that an EEG done three months after S.D.'s acute presentation could be used to support a finding of prior encephalitis. Resp. Ex. C at 5.

Regarding Dr. Wiznitzer's first criticism based on the lack of clinical coordination of drowsiness, when S.D. was brought to the ER on May 18, her mother reported that she was sleeping more than usual the past week. Pet. Ex. 2 at 8 ("[S.D.] has been sleeping much more than usual."). This observation was repeated in several places in the records. See, e.g., Pet. Ex. 2 at 13 ("Sleeping much more than usual . . . ."); see also Pet. Ex. 2 at 19 (noting S.D. "presented for evaluation of vomiting, irritability, fatigue"); Pet. Ex. 2 at 181 ("complaining of gait abnormality with clumsiness . . . as well as fatigue").

After discharge from the hospital, S.D. saw her pediatrician for follow up on May 27, 2016 due to lethargy. On July 21, 2016, she again had lethargy. On July 27, 2016, S.D. saw her neurologist, Dr. Shafrir. During that visit, S.D.'s mother reported that at the time of her prior hospitalization, S.D. acted "oblivious to her surroundings" and remained unsteady. Pet. Ex. 3 at 73-74. During physical examination, S.D. did not respond to her name, did not follow a finger, or make eye contact. Dr. Shafrir's impression was an "encephalitic process [] with acute ataxia, sleepiness[,] and vomiting with decrease[d] . . . mental status (according to mother's description)." Id. at 75.

Therefore, based on the interpretation of the EEG and the clinical correlation by Dr. Shafrir of brain dysfunction, as well as the opinions of Dr. Nahm, the undersigned finds preponderant evidence that S.D.'s abnormal EEGs findings are "consistent with encephalitis." 42 C.F.R. § 100.3(c)(3)(i)(B)(2). The fact that the studies were not done during S.D.'s acute presentation does not defeat Petitioner's Table claim. Instead, they show that S.D.'s condition was ongoing, and consistent with her clinical condition of encephalitis.

In summary, the undersigned finds preponderant evidence of CNS inflammation based on S.D.'s abnormal CSF with elevated WBC, abnormal EEG consistent with encephalitis, and abnormal MRI consistent with encephalitis.

For the reasons described above, the undersigned finds that Petitioner has proven by preponderant evidence that S.D. suffered an acute encephalitis as indicated by evidence of neurologic dysfunction plus evidence of an inflammatory process in the brain.

4. Exclusionary Criteria for Encephalitis

Next, there are exclusionary criteria for an encephalitis claim:

> [r]egardless of whether or not the specific cause of the underlying condition, systemic disease, or acute event (including an infectious organism) is known, encephalitis shall not be considered to be a condition set forth in the Table if it is shown that the encephalitis was caused by:
>
> (A) An underlying malignancy that led to a paraneoplastic encephalitis;
>
> (B) An infectious disease associated with encephalitis, including a bacterial, parasitic, fungal or viral illness (such as herpes viruses, adenovirus, enterovirus, West Nile Virus, or human immunodeficiency virus), which may be demonstrated by clinical signs and symptoms and need not be confirmed by culture or serologic testing; or

(C) Acute disseminated encephalomyelitis (ADEM). Although early ADEM may have laboratory and clinical characteristics similar to acute encephalitis, findings on MRI are distinct with ADEM displaying evidence of acute demyelination (scattered, focal, or multifocal areas of inflammation and demyelination within cerebral subcortical and deep cortical white matter; gray matter involvement may also be seen but is a minor component); or

(D) Other conditions or abnormalities that would explain the vaccine recipient's symptoms.

42 C.F.R. § 100.3(c)(3)(ii).

Here, there is no evidence that S.D. had an underlying malignancy. MRI studies did not show any lesions or tumors to suggest a malignancy. Further, there is no evidence to suggest that she had ADEM. Her MRI studies were not interpreted to show ADEM, and she was never given the diagnosis of ADEM.

Dr. Wiznitzer opined that S.D.'s acute cerebellar ataxia occurred about three weeks after a URI.[13] S.D.'s medical records reflect that prior to her visit on April 19, 2016, she had a URI, as reported by her parent. However, on that date, her fever had resolved and her physical examination was normal. She did not have redness or signs of infection of her ears, nose, or throat. On the date of vaccination, her physical examination was normal. There was no evidence of any infection. Similarly, the PCR tests and viral studies done showed no evidence of infection. While S.D. previously may have had a URI, there was no continuing evidence of infection.

Further, a possible infectious cause was considered during her hospitalization; however, testing did not reveal that infection was the cause of her encephalitis. S.D.'s CSF was tested by PCR for EBV, HSV, CMV, VZV, and enterovirus infections. See Pet. Ex. 2 at 32, 40, 49-51, 53. Additionally, S.D. did not present with a history of an antecedent infection or fever. ED physician Dr. Therese Canares opined that the "[l]ack of fever suggests low likelihood of infection." Pet. Ex. 2 at 11. When S.D. was seen by neurology on May 19, 2016, it was noted that she "did not have a viral illness prior to the gait abnormality." Pet. Ex. 2 at 22. The CSF was "possibly consistent with a viral process," but the PCR studies all came back negative. Id. at 177-78. S.D.'s CSF results were not consistent with a bacterial process. Id. at 172.

[13] Dr. Wiznitzer cites portions of the medical records to support his opinion that S.D. had an antecedent viral infection that caused her encephalitis. For example, he cites an entry in the medical records that stated S.D.'s condition was "most likely" related to a viral illness. Resp. Ex. A at 5 (citing Pet. Ex. 2 at 24). However, the same entry also contains the word "possible" in regard to the viral etiology. See Pet. Ex. 2 at 24 (noting the lumber puncture "shows a possible viral infection"); see also Pet. Ex. 2 at 29 ("Etiology of presentation remains uncertain at time of discharge, but work-up to date overall reassuring and appears possibly consistent with viral process."). Although the reference from the records uses the word "possible," Dr. Wiznitzer did not cite to that portion of the record. Thus, he may have failed to consider all the evidence.

On May 20, 2016, the neurology notes state "[c]oncern for likely acute cerebellar ataxia with negative workup to date." Pet. Ex. 2 at 178. Discharge summary states that CSF viral studies were negative and that the "[e]tiology of presentation remains uncertain" but "possibly consistent with viral process." Pet. Ex. 2 at 182.

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate something occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard); Moberly, 592 F.3d at 1322.

When taken as a whole, the entries by the physicians, which include the words "possible" and "most likely" together, as well as the word "possibly," the weight of the evidence does not rise to the standard of more likely than not. Further, given that the diagnostic testing failed to show any viral etiology, and the fact that all of S.D.'s physical examinations failed to show evidence of red ears and throat, cough, or any other evidence of viral infection, the undersigned finds that evidence that S.D. had a viral illness that caused her encephalitis is lacking.

For all these reasons, the undersigned finds that there is not preponderant evidence that S.D. had an alternative cause of her encephalitis or that she met any of the exclusionary criteria set forth in the Table.

5. Chronic Encephalopathy

Finally, Petitioner must show that S.D.'s "acute encephalitis . . . result[ed] in a chronic encephalopathy," which "occurs when a change in mental or neurologic status, first manifested during the applicable Table time period as an acute encephalopathy or encephalitis, persists for at least [six] months from the first symptom or manifestation of onset . . . of an acute [] encephalitis." 42 C.F.R. § 100.3(c)-(d).

The onset of S.D.'s acute encephalitis was approximately May 11, 2016. After her hospital discharge on May 20, 2016, she frequently presented for medical care and treatment of her condition. S.D. had an abnormal EEG on October 7, 2016, showing bitemporal cortical dysfunction, indicating a persisting post-encephalitic condition. Approximately one year later, in June 2017, a video EEG was abnormal and showing "intermittent high amplitude rhythmic slowing in the left than the right posterior quadrant, spreading to the left and the right temporal region." Pet. Ex. 3 at 23. Dr. Shafrir noted that "[S.D.] had significant encephalopathy following her recovery from acute cerebellar ataxia with excessive sleepiness and slowing on her EEG." Id. at 5.

Respondent asserts that S.D.'s acute cerebellar ataxia lasted less than four months, based on an assessment in August 2016 showing "age appropriate gross motor skills with no recommended physical therapy," and notes from a visit to Dr. Shafrir in August 2016 showing that her balance and gait were normal. Resp. Response at 21. However, these two observations do not negate the neurological abnormalities at the time of S.D.'s acute presentation, her abnormal MRI results, or abnormal EEG studies that showed abnormalities consistent with the

sequelae of encephalitis for a period of greater than six months.

Respondent's expert also asserts that S.D.'s ongoing "dysfunction" was the result of a pre-existing developmental disorder. Dr. Wiznitzer opines that S.D.'s "neurological dysfunction that persisted for more than [six] months was her autism spectrum disorder." Resp. Ex. C at 6.

At her four-month well-child visit, S.D. was noted to be developing normally. At her nine-month visit, there were no developmental concerns. At her one-year well-child visit, on April 27, 2016, the date of the MMR vaccination at issue, she was noted to be meeting her developmental milestones except those related to speech. At this visit she was assessed with a speech delay. There was no other evidence of developmental delay before S.D. suffered from acute encephalitis. The undersigned does not find that one assessment of speech delay in a child of one year of age constitutes preponderant evidence of developmental delay. Nor does it provide preponderant evidence of an autism spectrum disorder.

Regarding any later diagnosis of autism, or autism spectrum disorder, the undersigned finds that it has not been alleged to be vaccine related, and there is no evidence to support a vaccine-related cause. The undersigned's finding here relates only to a Table claim of encephalitis following the MMR vaccination. Autism has not been deemed a vaccine injury in the Program and the evidence here does not support such a finding that autism could be or was vaccine related. The undersigned does not treat any autism diagnosis S.D. may have had or now has as a compensable sequela of her encephalitis as it is unrelated to her vaccine injury. This Ruling finds Petitioner is awarded damages associated with only the effect of S.D.'s encephalitis.

Based on the medical records, the opinions of S.D.'s treating physicians, the EEG results, and the opinions of Dr. Nahm, there is not preponderant evidence that S.D. had a pre-existing development disorder or underlying autism spectrum disorder that caused or contributed to her acute encephalitis or her chronic encephalopathy arising from her acute encephalitis.

Therefore, the undersigned finds that Petitioner has proven by preponderant evidence that within five to 15 days of her MMR vaccination, S.D. suffered an acute encephalitis, and that it persisted for at least six months from the time of onset, thus meeting the Table definition of chronic encephalopathy.

V. CONCLUSION

For all of the reasons stated herein, Petitioner has proven by preponderant evidence that S.D. suffered from the Table injury of encephalitis caused by the MMR vaccination that she received on April 27, 2016. Therefore, Petitioner has established entitlement to compensation.

A separate damages order shall issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master